Understanding Psychopathology Notes

Understanding Psychopathology (Rijksuniversiteit Groningen)

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Understanding Psychopathology

Notes Week 1

1. Explain the critical features of “good” theories and why theories can be best seen as disposables (are not forever).
2. Explain why it is critical/important for therapists to know how and why their interventions work.
3. Describe and explain the arguments that have been raised against EMDR.
4. Explain the contribution of Marcel van den Hout’s experiments for our understanding of how EMDR might work.
5. Describe Clark’s cognitive model of panic disorder and explain how the various components of the model have been tested.
6. Explain how (and where) the various effective interventions for panic disorder tap in to Clark’s model.
7. Describe the network approach (as highlighted in the article of Richard McNally) and explain how this perspective differs from the traditional latent factor models of psychopathology.
8. Illustrate the network approach for panic disorder and major depression.

Cognitive Approach to Panic

Phenomenology of panic attacks:

- intense feeling of apprehension, sudden onset, associated with wide range of physical sensations
- Occur in feared/safe situations

Cognitive Model of Panic Attacks:

- Pharmacological and physiological agents may not have direct effects but may provoke panic only if the sensations they produce are interpreted as such
- Catastrophic misinterpretation of certain bodily sensations -- perceived as much more dangerous than they really are
- Panic attacks preceded by heightened anxiety
    - Anxiety in the form of anticipation of the attack
    - Anxiety unconnected to anticipating an attack
- panic attacks not preceded by anxiety
    - Trigger is perceiving a bodily sensation which is caused by a different emotional state as catastrophic

Panic Attack Research:

1. Ideational components of panic anxiety: Panic patients are significantly more likely to have thoughts concerned with anticipation of illness, death, or loss of control
2. Perceived sequence of events in panic attack: panic patients report that the first thing they notice is physical feeling

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3. Hyperventilation: voluntary hyperventilation can induce panic-like state, and hyperventilation accompanies naturally occurring panic attacks
4. Lactate induced panic: sodium lactate used to induce panic attacks -- occur more in panic patients, showing they have a tendency to interpret bodily sensations catastrophically
5. Effects of physiological treatment: CBT and behavioural approaches

    a. Brief voluntary hyperventilation
    b. Explain and discuss how hyperventilation induces panic -- during a panic attack patients may be over-breathing
    c. Train a pattern of slow controlled breathing
    d. Training in more appropriate cognitive responses to bodily symptoms
    e. Identify and modify panic triggers
    f. CO_2 inhalation therapy associated with significant reductions in panic attack frequency and fear of autonomic sensations

6. The role of biological factors in panic:

    a. contribute to triggering of an attack (patients experience more fluctuations in body state than others)
    b. Influence the extent to which a perceived threat produces an increase in bodily sensations
    c. Individuals may be more likely to experience sudden sympathetic activity surges -- more likely to interpret this as catastrophic

7. Effects of pharmacological treatment: imipramine seems to have the most anti-panic effect

    a. Treatments which fail to change tendency to interpret sensations as catastrophic will have higher rates of relapse than treatments which succeed in changing interpretations

EMDR - eye movements superior to beeps in taxing working memory and reducing vividness of recollections

Eye movements in EMDR during recall of aversive memories reduce their vividness and emotionality -- reconsolidation of memories are affected by experiences during recall

- Memories are blurred during and after eye movements (are labile during recall)
- Moving eyes horizontally and vertically is equally effective
- Same effects occur if WM is taxed with other secondary tasks - counting, drawing complex figure
- Negative images about future events can also be treated like this

Working memory:

- CE: central executive -- allocates and divides attention between tasks, activates memories, inhibits distractors
- Visuospatial sketchpad: processing visuospatial info

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    - Eye movements reduced image vividness by temporarily disrupting active maintenance and manipulation of traumatic images in the VSSP of WM

- Phonological loop: processes verbal info

Binaural stimulation:

- Patient wear a headphone and hears beeps alternating Left-Right
- Does not require person to actively engage in cognitive or motor operations - how does it tax WM?
- About 50% of sessions

Discussion:

- Data from the experiments suggest that eye movements affect memory by taxing the CE during recall, binaural stimulation taxes the CE to some degree (0.25 that of eye movement)
- Effects of binaural stimulation are ⅓ that of EMDR

Retrieving and Modifying Traumatic Memories: Recent Research Relevant to Three Controversies

Controversies Concerning Memories of Trauma

- Repression perspective: people become incapable of recalling childhood abuse because of how emotionally traumatic they are
- False-memory perspective: people who report recovering memories of abuse are often mistaken
- Traumatic memories are about as coherent as positive memories -- fragmentation reflects a persons style of recounting memories rather than something related to the trauma
- EMDR eye movements reduce memory vividness and emotional intensity ratings after the intervention (the precise mechanism mediating the effects of eye movements on memory remains unknown)

Study shows: Some children can expereince childhood sexual abuse without understading it as abuse -- without experiencing the terror aspect of it (can suffer PTSD when recallingit later)

Can Network Analysis Transform Psychopathology?

2 approaches attempt to explain how psychiatric symptoms do not co-occur randomly, some are more likely to co-occur than others

- Categorical approach: discrete entity is common cause of symptoms emergence and covariance (co-occurence of insomnia, sadness, etc is depression)
- Dimensional model: underlying cause of symptoms emergence and coherence (symptoms covariation is caused by common factor)

Network Approach

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- Symptom covariance emerges from dynamic, causal interactions among symptoms (possibly self-reinforcing)
- Symptoms are not reflective of underlying mental disorders, they are constitutive of them
- *Key concepts:*
    - Nodes (objects of study) and edges (connections between nodes)
        - Weighted edge: magnitude of the connection (represented by thickness of the edge)
        - Unweighted edge: two symptoms are connected
        - Directed edge: arrows pointing in the direction of prediction, and causation
        - Undirected edge: single lines that connect paris of symptoms
    - Node centrality
        - High central nodes are those of greatest importance to the network
        - *Degree centrality*: number of edges connected to it
        - *Strength centrality:* (in weighted networks) thickness = magnitude of association, and thus the probability that the activation of one node will be associated with activation of nodes connected to it
            - Can have in strength or out-strength centrality depending on the direction of the edges
        - *Expected influence:* takes into account both negative and positive edges (more positive edges = higher strength)
        - *Closeness centrality:* average distance from a node to all other nodes in the network
        - *Betweenness centrality:* number of times that node lies on the shortest path between two other nodes (can be bridge between two disorders)
    - Types of Networks
        - *Association networks:* nodes with stronger correlations are placed at the center, and weaker on the outskirts -- looking at strength of association between two symptoms
        - *Partial correlation networks:* helps to discern causal relations (compute correlation between two symptoms after adjusting for the influence of all other symptoms in the network
        - *Relative importance networks:* each edge depicts the relative importance of a symptoms as a predictor of another symptoms (weighted and directed)
        - *Bayesian networks:* produces directed acyclic graphs (best fit model)
            - Treating a symptom that occurs in the DAG model should turn of activation in its descendant symptoms

<u>Lecture 1 Notes</u>
Theories and Psychopathology - why so important?

- Connects the dots of loose facts/observations

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- Guides further inquiries
- Series of coherent hypotheses or propositions about one phenomenon/several phenomena (can not know for sure if it is true)
- Understanding the *origin* of psychopathology
- Understanding factors involved in *persistence* of psychopathology

Theory must be: consistent with known facts, testable, tested and showing predictive validity, internally consistent, as simple as possible, the fewer the assumptions, the better

- Complementary theories (work together): social anxiety disorder has genetic, developmental, and learning aspect
- Convertible: gen x development x learning experience together = higher risk
- Incompatible? Then only one is the best

The case of the panic disorder:

- Psychiatric CO2 theory
- Hyperventilation theory
- Need a theory that can cope with findings that both an increase and reduction of CO2 -> panic attack
- *Cognitive approach:* stimulus gives rise to physical sensation, and if individual has a catastrophic misinterpretation of this they can perceive a threat ➔ self reinforcing cycle that becomes a panic attack

    - bag/knob gives people control, they can prevent that PA escalates

Theories are not forever:

- Original psycho-analytic explanation of phobias: little Hans' fear of horses (based on Freudal assumptions)

    - Intelligent unconsciousness - testable?
    - Innate incestuous tendencies - at the time maintstream
    - Predictive value: fear disappeared after father discussed the issue with Hans (is this proof, or the spontaneous course of fears?)

- No theory ➔ rash empiricism (je probeert eens wat)

    - ECT or antidepressant meds for panic disorder

Depression a (brain) disease?

- Metaphor that depression is like diabetes is used
- Depression connected to many symptoms -- does it give rise to all these? Or are there other factors that give rise to them? Symptoms can lead to other symptoms
- Not necessarily caused by brain dysfunction

Sometimes theories are defective: psychosis theories often only look at genetics but we should see how similar our own lives are

- Trauma -- "recovered memories"

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Need theories to be starting point for interventions, even if defective

Optimal interventions are techniques derived from optimal theories, without optimal theories, therapy is hocus pocus, beware of pragmatists who do not want to know how theories work

Notes Week 2

1. indicate and explain the *implicit assumptions* of psychopathology and some of the *drawbacks* of the DSM(5);
2. indicate the uniqueness and value of the *developmental (process) perspective* on psychopathology, as discussed during the lecture and in the literature;
3. indicate the uniqueness and value of the *complex systems approach* to psychopathology, as discussed during the lecture and in the literature;
4. explain the concept of *ergodicity*, that is, indicate its main properties and features;
5. indicate the meaning of *ergodicity* for psychology in general and for psychopathology in particular, and apply it to a concrete example;
6. explain the concept of *network*, that is, indicate its main properties and features;
7. indicate the role of symptoms (criteria) and disorders (diagnoses) in the understanding of comorbidity within the *network model of psychiatric symptoms*.

The New Person-Specific Paradigm in Psychology

Ergodic theorems: mathematical-statistical, motivated by problems of statistical physics

Conditions for Ergodicity

- Combines interindividual with intraindividual
    - 3D data box: time, psychological variables, and subjects as dimensions
- Homogeneity:
    - Statistical model should apply to all subjects in the population
    - Factors and factor loadings must be invariant across subjects
    - Factor model of population = R technique
    - Factor model of individual subject = P technique
- Stationarity:
    - Data must be stationary - constant statistical parameters over time

Discussion

- Psychological processes like cog info processing, perception, emotion, and motor behaviour occur in real time at the level of individual persons
    - Person specific variables
    - Should analyse using intraindividual variation

The Small World of Psychopathology

1. Half the symptoms in DSM-4 are connected

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2. Architecture of the connections conforms to small world structure: high degree of clustering but short average path length
3. Distances between disorders in structure predict comorbidity rates

Symptoms patterns in comorbid disorders shows causal networks
- There is not just one source of comorbidity
- Most common: Major Depressive Episode and Generalized Anxiety Disorder

Network approach: symptoms are not viewed as indicators of latent conditions, but as components in a network
- Comorbidity - results from direct relations between symptoms of multiple disorders
- Significant amount of comorbidity arises from bridge symptoms
- Construction of network:
    - Each DSM-4 symptom = node
    - Draw edges between any two nodes if they are criteria of the same disorder
    - Roughly half of the symptoms are connected
    - Centrality of nodes:
        - Degree: number of connections a node has with other nodes (this network is a “single-scale” network)
            - Symptom with highest degree: insomnia
        - Betweenness: probability that a node lies on a path between two other nodes
            - Highest: irritable, distracted, anxious, depressed
        - There are many short roads to comorbidity
- Small world property
    - Implies that symptom activation will spread rapidly through the network
    - Path lengths and empirical comorbidity rates are correlated as would be expected if symptoms activation propagates through network structure
    - For a given symptoms, the probability of occurrence increases monotonically with the number of neighbouring symptoms that are activated

Discussion
- Comorbidity explained through genetics, neuroscience, etiology of mental disorders
- Strength of symptom connections may be genetically controlled
- Mental disorders are unlikely to correspond to a single homogeneous neural substrate
- Network model yields plausible explanations for patterns in data, and illuminates the limited success of other research paths
    - Psychosystems approach - investigate inherent complexity of mental disorder

Complex Systems Approach to the study of change in psychotherapy
Presenting basic principles of complex systems

General Principles of change in complex adaptive systems
- Interconnected traits that evolve and self organize into functional units: *attractor states*
- Adaptive system is flexible as conditions change, but can maintain functional integrity
- Developed attractor patterns are relatively insensitive to challenges/new info
- Tipping points: point where dominant state can suddenly shift (abrupt)

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        - Knowing when this will occur can have important implications
            - Warning signs: critical fluctuations (rising variability that indicates system dynamics are breaking down), critical slowing (slower recovery/ return to baseline)
        - There can be a period of flickering between attractors - until one state is strong enough to capture the system (new exercise routine example)
        - Components and processes can operate on different time scales
- Network destabilization and transition model
    - Psychopathology is an attractor state with interacting elements of cognition, emotions, behaviour, physiology
        - Contextual factors also highly important
    - Patterns of healthy/psychopathological networks can be established
    - Change in psychotherapy:
        - Make minor adjustments to maladaptive patterns
            - Harm reduction strategies
        - Switch from a pathological to healthier attractor (must be an alternate to switch to)
            - Positive emotion activation approaches
        - Destabilize the pathological attractor and develop healthier patterns of functioning
            - Exposure therapy, cognitive restructuring
            - Characterized by system-wide disturbance
        - Weakened pathological network can still be activated by stressful life events of random forces
            - Need to enhance inhibition of switching back

<u>Continuities and discontinuities in psychopathology between childhood and adult life</u>

Childhood origins of schizophrenia

- More likely to have social, emotional, behavioural problems as kids
- Have stable pattern of impaired attention
- Delays in early motor development, impairments of receptive lang and cog functioning
- Shows continuity of psychotic-like symptoms from childhood to adulthood
- Can't predict who of those in high risk group will actually develop schizophrenia
- How do precursors turn into overt schizophrenia?
    - Developmental changes in brain during late adolescence
    - Heavy early use of cannabis in adolescence increases risk
    - Certain types of social adversity (migration, isolation) might contribute

Neurodevelopmental disorders

1. Manifest by delay in maturation
2. Not marked by the remissions and relapses that are characteristic of most other disorders
3. General tendency for impairment to lessen with age
4. Disorders all involve some degree of cognitive impairment
5. Tendency for neurodevelopment disorders to overlap
6. Genetic influences and individual differences are strong
7. Environmental influences probably also contribute
8. Disorders all show marked male majority

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- *Autism spectrum*
    - Start showing abnormalities at 18 months
    - IQ lower than 50 from childhood -- bad prognosis (main interest in those with IQ over 50)
    - Best outcomes for group who had IQ of at least 70 (but still not many)
    - Variations in IQ above 70 were of negligible prognostic importance
- *Specific Language Impairment*
    - Can gain reasonable level of communicative language, reading and spelling stay impaired
    - Only ⅛ employed
    - The disorder shows far more than the end of a normal continuum (affects many children)
    - Genetic influences greater in persistent impairments
    - The deficit is social as much as linguistic
- *Dyslexia*
    - cognitive/language deficits are represent in early years of life, but not distinctive enough to be used for diagnosis
    - High proportions of adults who showed disability at school age now still struggle, but no increase in adult mental disorders
- *ADHD*
    - Originates in cognitive deficit/bias
    - Executive dysfunction and delay aversion
    - Poses problem by age of 3
    - Adult psychopathology more frequent in those who had ADHD from childhood
        - Childhood ADHD is precursor for later antisocial disorder
    - Marked drop between adolescence and adulthood in proportion of individuals who meet ADHD criteria
    - ADHD features function dimensionally, not categorically

Depression

- Adolescent onset is associated with direct strong risk for recurrence in adulthood
    - 40-70% get MDD recurrence
- Recurrence patterns do not hold for depression that was first found before puberty
    - Childhood onset and adolescent onset are qualitatively different
- Genetics: transmission of depression is linked with earlier age of onset, greater risk of recurrent MDD, and greater associated impairment
    - Environment: high amount of negative life events also adds to it
- Cognitive vulnerabilities - as early as 5 children can have depressogenic cognitive biases
    - Degree of stability from childhood to adulthood is unknown
- *Sequential Comorbidity*
    - Anxiety starts in childhood, precedes onset of adolescent MDD
    - Anxiety could be age-dependent expression of depression
        - Separation anxiety, generalized anxiety and panic most likely to develop MDD
    - Adults with GAD highly likely to have had MDD in childhood/adolescence
    - Progression from substance abuse to MDD - causal mechanism?

Antisocial Behaviour

- *Heterogeneity in Outcome*

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    - Childhood onset antisocial behaviour: associated with neurodevelopmental difficulties and adverse rearing conditions ➔ poor outcomes, longterm for majority
    - Adolescent onset antisocial behaviour: more normative, persistence is not expected to last beyond end of puberty
- *Mechanisms for persistence and desistance*
    - Highly heritable
    - Highly environmental
        - Genes may make individual more vulnerable to environment
    - Unclear whether age at onset per se contributes to variations in persistence/intervening mechanisms
- *Risks for Psychiatric Disorder in Adult Life*
    - Childhood and adolescent conduct problems strongly associated with increased risk for psychiatric disorders later in life
    - Conduct problems are strong predictors for substance abuse problems
    - Alcohol dependence in adolescence also “ensnares” young men into more persistent antisocial behavior
    - By the teens comorbidity between depression and conduct/oppositional defiant disorders are high

Substance Abuse

- Childhood and adolescent conduct disorder is linked to initiation and progression of substance abuse
- Later adolescent substance abuse increases risk for persistence in crime
- Depressed mood is strongly associated with use of substances - reflect self medication efforts?
    - Substance abuse does predict later depression
- Genetics are stronger for abuse and dependence than for initiation of drug use
- Early initiation leads to progression in substance abuse and difficulties in later functioning

Overarching Themes

- *Heterotypic continuity and psychopathological progression*
    - Heterotypic: disorder remains the same but manifestations have altered
    - Meaningful continuity in the course of a disorder but the ways in which it manifests itself changes with age (early anxiety and later depressive disorders)
    - Obvious evident continuity in some impairments (reading and spelling)
    - Pure language disorder does not exist - is usually a broader cognitive deficit that includes social cognition
- *Early age at onset*
    - Early age is often assumed to be direct causal factor ➔ but this should not be assumed without looking for alternative explanations
    - Are differentiations due to puberty or chronological age?
- *Mediators of continuities and discontinuities in psychopathology*
    - Genetics (at least in party), creates susceptibility to environmental factors
    - Kindling effects - experience of a mental disorder brings changes that make recurrence more likely
    - Antisocial psychopathology increases likelihood of exposure to risk environments in adult life

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    - Adverse experiences in childhood and adult life may influence likelihood of depression
    - Ways in which individuals respond to psychopathologies can increase/decrease later risks
    - Indications that how people conceptualise what is happening to them influences the course of what they are suffering
- *How early in life is behaviour predictive of later psychopathology?*
    - Does later psychopathology involve abnormalities that are present at early age?
    - Can such abnormalities be measured reliably and validly to provide useful individual prediction?

Theoretical Perspectives on Developmental Pathways

- Development includes continuity and change, with both including coherence, lawfulness, and organization
- Most models note importance of personal agency, and placing emphasis on the mediating role of emotional/behavioural regulation

Developmental Systems and Psychopathology

- Contradictions found in the labels we use to divide and categorize psychological phenomena and the dynamic reality
- Contradiction between abstracted diagnostic schemes used to categorize individuals and the complex dynamic processes of individuals themselves
- Contrast between study of serious mental health disorders and mental health -- may be that study of each is required for the understanding of the other
- Need to study both nature and nurture to understand the other better

Dominant Issues in the Field

- How to define pathology?
- How to understand individuals and their development?
- How do we conceptualize the environment?
- Traditional medical model doesn't work for developmental psychopathology as it assuems
    - Same entity will cause the same disorder in all affected individuals
    - Same symptoms at different ages are caused by same entity
    - Specific disorder of children should lead to similar adult disorders

Developmental Psychopathology

- Continuity: severe symptoms are placed on same dimension as more normal behaviours
    - However, pathologists categorize discontinuity: abnormal is different from normal (view of humans as determined by their biology)
- Achievements in lab are not readily translatable into achievements in society at large, nor does scientific reason directly change social behaviour
- ➔ need a model that matches complexity of human behaviour
- Big research done on schizophrenic parents/children shows that nature of environment is as important as biological heritage for future mental health, and that symptoms are different between children and adults

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Conceptualizing Pathology

- What does it mean to be disordered? Are disordered children different in kind of degree?
- Mental retardation example:
    - 2 types: kids that are 2 st. dev. below the mean (including the 6-hour retardation, when difficulty is only shown when looking at scholastic standards)
    - Second type is organically impaired, and therefore has low IQ scores (different from those who are only at the low end of normal)
- Do the children with whom we are concerned represent the extreme of a normal distribution, or are they different in kind from the rest of the population?

Conceptualizing Individual Development

- Using operational definitions ➜ dividing world into categories, catering to reductionist tendencies to view behaviour as traitlike characteristics of the behaver
- *Pattern of adaptation:* when situations change the individual changes, categories are not inherent in individual but in relationships between individuals and situations
    - Very complicated to know everything about every situation
- *Traits:* elaborate schema with clear categories and descriptions and criteria for classifying children
    - Depression:
        - There are 3 levels: depressed mood, depressive syndromes, depressive disorders
        - Rare for children who have depression problems to only have depression, often combined with emotional and behaviour problems (comorbidity)
    - Conduct disorder:
        - Early authority conflict pathway characterized by stubborn behaviour, defiance, authority avoidance
        - Covert pathway - property damage, moderate to serious forms of delinquency
        - Overt pathway - aggression, fighting, violence
        - Comorbidity with attention deficit, substance abuse

Conceptualizing the Environment

- Social circumstances are more powerful risk factor than any of the parental mental illness measures
- 10 environmental risk factors: history of maternal mental illness, high maternal anxiety, parental perspectives that reflect rigidity in attitudes towards child's development, few positive maternal interactions with child during infancy, head of household in unskilled occupations, minimal maternal education, disadvantaged minority status, single parenthood, stressful life events, large family size
- Are there protective factors? Or simply positive pole of risk factors (promotive factors)
- Multi Pathology: associated with a plenitude of risk factors and paucity of promotive ones
- *Environtypes:*
    - Child’s behaviour at any point is a product between the phenotype (child) the environment, and the genotype

Lecture 2: Life Span and Psychopathology

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Developmental Psychopathology (DPP)

SOME ISSUES IN THEORY BUILDING

Developmental Psychopathology

- Study of developmental processes underlying psychopathology
    - Studies formation of condition
    - Prevention of condition
    - And maintenance of a specific development
- Influence of development on expression and properties of psychopathology
    - Is child depression same as adult depression
- Influence of psychopathology on rest of development
    - What is necessary for typical/desirable trajectory

Different Approaches

- Categorical: classification (all or none)
    - Limitations: no attention for developmental aspects (independent of age)
    - Subjective distress and impairment of functioning are not applicable to children - so how can we use DSM for kids?
        - Need to look at whether a behaviour or symptoms interferes with child's capacity to continue developing
    - Application of adult classification system for pathology ignores specific problem of children, and interpersonal context and family setting *(contextual)*, children's development can go in many directions *(plasticity),* comorbidity in children is about 50% *(complex)*
- Dimensional: on a line (more or less)
    - Limitations: accounts for only a little for different manifestations/forms of psychopathology at different ages
        - Does not deal with development much either
    - Doesn't account for contextual differences
- Process approach: change (process laws)
    - Specify the causal mechanisms by which risk and protective factors lead to specific individual developmental outcomes
    - How are things actually happening?
    - Change phenomena (gradual)
        - Development
        - Acquisition
        - Learning
        - Growth
        - Etc.
    - Transition phenomena (abrupt)
        - Changing your mind
        - Insight
        - Relapse after therapy
        - Identity switch

Process Laws and Psychopathology

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1. Every step depends on the previous
    a. Possible external factor depends on the momentary state of the process
    b. Looks for equilibrium - can change in itself, you don't need a specific outside factor for there to be change
2. There are feedback loops within the system
    a. Positive ➜ strengthens a process ➜ stabilizing patterns
    b. Negative ➜ weakens a process ➜ destabilizing patterns
    c. Two subprocesses (or symptoms) can also influence each other
3. Coupling leads to attractors
    a. An attractor = relatively stable and self- organizing state of the system
4. System and Environment influence each other
    a. Selection of environment (selecting friends)
    b. Direct influence (aggression leads to aggression)
    c. Attitude towards environment (distrust)
    d. Environtype, phenotype, and genotype ➜ development is a relational, coactive process
5. Processes depend on multiple time scales
    a. Long term: developmental processes
        i. Changes in environment, body and brain due to maturation and growth
        ii. Developed preferences, skills, traits, emotions
    b. Short term: behaviour
        i. Shaped momentary social, material and here and now
    c. These time scales influence each other

Ergodicity

- What is real?

Sample and Process

- Psychologists aim for relations between variables and assume that these variables and relations refer to what is going on in an individual, and are constant over time
- Relation between does variables does not mean causation

Realistic behavioural and cognitive systems are complex and non-ergodic!

- Statistical analysis of a few measurements of a large group and many measurements of one individual will not be the same

Importance of Ergodicity:

- Why should we assume that traits etc stay the same over time and only measure large groups once instead of individuals over a long period of time?
- DSM are based on implicit assumption of ergodic psychological systems, but that is wrong
- Criteria for Ergodicity: Homogeneity, Stationarity
    - Big 5 are stable at group level, but not at individual level

Role of Implicit Assumptions

Classical World View of Pathology

- Health and illness are to categorically different states of organism
- Pathogenic influence without sufficient counter influence leads to illness: can be reversed through medicine therapy to go back to health

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- Categorical differences, show there is underlying essence of problem, can test for the disorder, can cure that essence

Types of Causality:
- Monocausality: one event causes another event, which causes another
    - Traceable root cause
    - Newtonian worldview
- Multicausality: no single cause, all components, processes, events, interactions are relevant
    - Holistic worldview

Symptoms, Comorbidity (small world)
- In DSM, symptoms are passive indicators of latent conditions
    - Disorders have the power to create symptoms
    - So comorbidity has one root cause

Comorbidity:
- Symptoms are not indicators of latent disorder, rather they are complaints that cause are are related to other symptoms
- Comorbidity is an individual developmental process
    - Results from concrete and direct relations between symptoms
- Symptoms are primary and form a network
- *Symptom space*: network of all DSM-4 symptoms, drawn as nodes- symptoms that belong together are connected by edges
- *Bridge symptoms*: connect symptoms of different disorders by (indirect) path

Small world: everyone is six human introductions away from any other person in the world

Small world networks: connections between nodes are short
- Clustering coefficient is high
- Average shortest path is low
- Many hubs of nodes

Notes Week 3
- Discuss executive functions (EF) on the background of the unity-diversity model
- Describe the relation of EF impairments to psychopathology and treatment success
- Explain the relevance and implication for assessment and treatment

Advancing Understanding of Executive Function Impairments and Psychopathology: Bridging the gap between clinical and cognitive approaches

EF is comprised of a set of control processes, mainly supported by prefrontal cortex
- EF deficits may be risk factors for emotional, behavioural, psychotic disorders

EF Impairments Associated with Psychopathology

Impairments on Specific EF Components: Inhibition, Shifting, Updating, Working Memory
- Largest EF deficits found for individuals with schizophrenia
- These tasks also impaired in mood disorders

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        - Those with MDD/OCD are significantly impaired, with small to medium effect sizes in shifting, inhibition, updating, and WM (only MDD)
        - Bipolar disorder slightly more impaired, with medium effect sizes in shifting etc
- PTSD also effects EF
    - Or might be co-occurring depression
- Trait anxiety, specifically worry, is associated with impairments in inhibiting competing responses part of EF
- ADHD associated with impairments in shifting, etc, and visuospatial WM and verbal WM manipulation
    - also impaired in externalizing disorders: ODD, CD (can be co-occurring ADHD)
- Substance use
    - Shifting, inhibition, WM impairments
    - Poor EF may be a risk factor for other substance abuse issues

Complex Tasks: Verbal Fluency and Planning
- Deficits in semantic verbal fluency task high in many disorders (highest in MDD, Schizophrenia)
    - Shifting among subcategories, monitoring for repeated words, memory retrieval impairments

Limitations and Suggestions for Future Research
Models of EF
- Individual differences in EF show both unity and diversity
- Behavioural level:
    - Focused on partly overlapping sets of EF components
        - Central executive system containing subsystems for coordinating performance between tasks, resolving interferences from dirstractors, switching between tasks, interfacing with long term memory
        - WM, cognitive flexibility - core aspects of EF?
        - Top down/lower level processes and monitoring processes

    neural level:
    - Distinct but interconnected prefrontal regions support functions such as setting task goals, initiating responses, monitoring performance,

Unity/Diversity model
- Updating WM
- Shifting
- Inhibition
- Common EF and shifting-specific components sometimes show opposing patterns of correlations with other measures
    - Rumination: better performance on goal maintenance, but worse performance on task requiring fast shifting
- Different components predict individual differences in clinically important behaviours

** measuring EF often involves task impurity, meaning that other factors and processes will also be involved, not just EF task you want to measure

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Transdiagnostic Mechanisms of Psychopathology in Youth: Executive Functions, Dependent Stress, Rumination

Unity/Diversity Model of EF

- *Shifting:* switching between task sets or response rules (classifying by shape or colour)
- *Inhibition:* suppressing or resisting automatic responses in order to make less automatic but task relevant responses (Stroop task)
- *Updating:* monitoring for and adding task relevant info to WM, deleting no longer relevant info
- *Common EF:* ability to monitor for and maintain goal and context info and use that info to bias ongoing processes

Bifactor Models of Psychopathology and Links to EF

- Psychopathology best conceptualized as continuous symptom dimensions
- Executive dysfunction is a risk factor for common psychopathology
    - P factor, specific internalizing and externalizing dimensions are associated with poorer EF in youth

Mechanisms EF and Psychopathology

- Poor EF can lead to stress generation
- Link between EF task performance and internalizing symptoms increases with age from puberty to adulthood
    - Adult caretakers compensate for younger adults poor EF (reminders to do homework), which prevents that EF to become stressful life events
    - Dependent stressful life events strongly predict rumination
        - A transdiagnostic risk factor

Current Study

- Tests association between latent unity/diversity model and latent dimensions of psychopathology liability in sample of youth
- Hypothesize that poorer common EF associated with common psychopathology liability
- (graphs and methods in paper)

Discussion

- For older youth, poorer common EF but not updating/shifting-specific factors was associated with higher internalizing liability
- Poorer common EF associated with more dependent stressful life events in older youth
- All youth: rumination and dependent stressful life events are associated with higher internalizing liability
- Results show that mediations between different forms of psychopathology and measures of different aspects of EF may be more explained by link between common EF and common psychopathology liability (p factor) and internalizing specific liability
    - Stress and rumination are mediators of these relations

Worse Baseline EF is associated with dropout and poorer response to trauma treatment for veterans with PTSD and comorbid TBI

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- Comorbidity of PTSD and history of TBI can complicate the course of recovery
    - Can exacerbate the cognitive/emotional dysfunction observed in each of these conditions alone
    - CBT is one of most effective PTSD treatments, can also help with mild TBI
    - OEF/OIF Veterans are more likely to have higher dropout rates
    - EF problems can be barrier in individuals trying to recover from PTSD/TBI
        - Bidirectional relationship
- Do neuropsych measures of EF associate with dropout and response to CPT in veterans with PTSD and TBI
- Results of study:
    - Poorer performance on baseline measures of EF is associated with dropout and reduced responsivity to treatment in veterans with PTSD and TBI
        - Can also be due to difficulties planning/problem solving how to get to therapy
        - EF problems can lead to difficulty maintaining motivation
    - Worse EF baseline performances are associated with less response to CPT -- less reduction in PTSD symptoms
    - Therapy techniques often rely heavily on executive processes to inhibit trauma related thoughts
    - Worse EF at baseline is associated with CBT dropout for substance dependence, GAD, reduced response to CPT in OCD, schizophrenia, GAD

Association between EF and Psychopathology: General or Specific?

- Which EFs are impaired in each disorder?
    - EF dysfunctions are usually widespread, severe, and chronic
- Poorer performance on several EF tasks is associated with general psychopathology factor (p factor)
- EF impairments may be more strongly associated with severity and chronicity of psychiatric problems than with distinct diagnoses
- Are impairments in general EF or in specific EFs related to psychopathology?

Discussion:

- Psychopathology and EF modeled as bi-factor models
- Shows impairments in multiple specific EFs are associated with general psychopathology - also present in specific problem domains
- P factor: captures all the shared variance between different problem domains - reflects cross domain severity and chronicity
- ASD and ADHD domains tested

Specificity of EF and Processing Speed Problems in Common Psychopathology

EF and processing speed in relation to multiple disorders

EF: top-down, goal directed cognitive processing

- Problem solving, impulse control, emotion regulation
- Key abilities: set-shifting and maintenance, interference control, response inhibition, working memory

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- Appears to be underlying shared mechanism that contributes to performance across different EF tasks
- Unity and diversity → can examine from holistic perspective and component perspective

Output Speed: composite of processes that generate speed of response on a task
- Contains speed of perception, efficiency of information processing and accumulation, speed of and bias toward response preparation, response execution
- Processing speed related to IQ and health and psychopathology

Selection of Psychopathologies
- ADHD
- Antisocial personality disorder (ASPD)
- Alcoholism, substance use disorder
- Depression
- Anxiety disorders
- These disorders all frequently co-occur

Speed, EF, and structure of psychopathology
- Speed accounts for some of the EF effects, because they are often confounded with speed ?
- *Specificity model:* different disorders are associated with different types of EF deficits
    - Considers EF as components
- *Severity model:* EF or speed impairment are related to overall severity of psychopathology rather than specific form of it
    - 2 proxies for severity: number of co-occurring disorders, clinician rating of Global Assessment of Functioning
- *Dimension:* EF and speed deficits are related to one or more shared, underlying psychopathology liability dimensions rather than specific disorders
    - Internalizing: related to depression and anxiety
    - Externalizing: related to anti-social and substance abuse
    - Other factors: general “p” factor

Tests Used in the Samples
- *Set Shifting:* used the Trail Making Test
    - Number circles in sequential order as fast as possible
    - Switch between numbers and letters in alphabetic-numerical order
- *Interference control:* stroop colour-word test
    - Read the name of the colour, Name the colour ink of XXXX, name the colour when ink and word are in conflict
- *Set maintenance and working memory:* Wisconsin Card Sorting Test
    - Deduce how to sort cards
- *Response inhibition:* Go-Stop Task

Discussion:
- Speed and EF were able to be differentiated and show distinct correlational patterns
- There may be developmental variation in the emergence of EF deficits
    - ADHD had strongest and broadest relationship to EF deficits
- Slow speed is robust correlate of psychopathology

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- Phenotypic structure:
    - Speed is intermediate phenotype for both externalizing and overall psychopathology severity
    - EF components are intermediate phenotypes for subsets of that liability

Lecture 3: EF and Psychopathology

Foundations of EF

- When encountering someone with a mental disorder, can you expect EF problems? YES
    - Mental disorders have small (OCD) to large (schizophrenia) EF effect sizes
        - Major depression has small to medium EF impairment for all EF problems
    - EF problems: shifting, inhibition, updating, visuospatial WM, verbal manipulation, verbal WM maintenance, semantic verbal fluency

- What are EF?
    - Main case: Phineas Gage after lesion had trouble with planning and other higher order cognitive tasks
    - EF: set of different cognitive control processes that enable regulation of lower level processes (top-down to achieve a goal)
        - In a new situation you need it: reasoning, problem solving, planning
        - Cognitive control: working memory updating, conflict monitoring, inhibition, set shifting
        - How successfully do we function in everyday life?
        - EF can predict treatment outcome
    - Models:
        - Data driven approach of conceptualizing EF
        - *Correlated factors model:* shared but distinct functions
        - *Nested Bi-factorial model*: there are some common EF, and specific functions
        - *Uni-factorial model*: there is one general common EF, no specific

- How to best measure EF?
    - Tower of hanoi: problem solving, visual cognition
    - Go - no go task: motor inhibition
    - N-back task: memory

  Task impurity problem:
  Specific EF factor + common EF factor + non-EF processes Error = Score on EF task
  (in increasing order of weight)

    - How to solve this?
        - Multiple measures targeting the specific EF aspect
        - Task selection are very specific to EF functioning (take long, there is no norm data)
            - test batteries (CANTAB)
            - traditional/clinical neuropsych EF measures (Tower of Hanoi)
            - Self reports (brief A) -- how do you experience problems in everyday life?

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Associations of EF and Psychopathology

- Are there core EFs that are associated with specific mental disorders?

- Severity model - EFs related to general psychopathology
- Specificity model - different disorders are associated with proglie of EF impairments
- Dimensional approaches:
    - Bi-factorial model of psychopathology: HiTOP
        - p (general psychopathology variable underlying all disorders)
        - internalizing (MDE)
        - Externalizing (substance abuse etc)
        - Explains why some disorders go together
        - Dimensional and categorical
    - Research Domain Criteria RDoC
        - 5 domains of functioning
        - Biological perspective
        - Dimensional

- Causal or correlated EF and psychopathology?
    - Mediation model:
        - genetics lead to EF problems, which increase probability of psychopathology
        - Or genetics lead to disorders, which then also causes EF problems
    - Liability index model:
        - Genetic predispositions, lead to both EF and psychopathology problems but these are correlated not causing each other

Notes Week 4

Objectives theme 4: Associative learning and psychopathology

After reading the articles and studying the information provided during the lecture

1. students can explain/define the following concepts:

Classical conditioning, UCS, UCR, CS, CR, Cue-reactivity, CCR (conditioned compensatory response), cue-exposure, D-cycloserine, CS+/CS- (differential conditioning paradigm), latent inhibition, sensory pre-conditioning, UCS revaluation (UCS-inflation), extinction, renewal, reinstatement, retrieval cues, evaluative conditioning (affective learning), referential vs. sequential (predictive) relationships, contra (counter) –conditioning, covariation bias, UCS expectancy bias.

In addition, students can explain/describe how these concepts can be measured/tested in the lab (and have been tested in empirical research).

2. Students can use/apply these concepts to explain:

a. why addiction is typically very persistent (idem binge eating problems).

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b. what type of intervention they could design on the basis of the presented models/evidence in the context of anxiety disorders, addiction, and bulimia nervosa, and how to explain the relevant components of these interventions in learning theoretical terms.

c. how phobic fears may develop and how it can be explained that some individuals do not develop a phobia in spite of having been exposed to a US experience, whereas others develop a phobia in the absence of contingent US experiences.

d. what type of learning mechanisms can help explain the persistence of anxiety and addiction problems.

e. why predictive relations do, but affective relations do not extinguish following exposure interventions (or CS-only extinction procedures).

f. how conditioning mechanisms can explain the frequent relapse of phobias and addiction following initially successful therapy.

g. what types of starting points can be derived from these explanations to improve the efficacy of currently available interventions.

Classical conditioning and the Acquisition of Human Fears and Phobias: review and synthesis of literature

Conditioning Processes in Humans

CS = conditioned stimulus
UCS = unconditioned stimulus
CR = conditioned response

- Only subjects who can verbalize the CS-UCS relationship show conditioning
    - This means there is some cognitive relational learning involved in processing this relationship
- Processes of UCS recalculation have importance in modulation of human CR strength; UCS revaluation can be achieved through:
    - Direct experience with UCS alone (may inflate/deflate the aversiveness of the UCS)
    - socially/verbally transmitted info about the UCS
    - Reaction of subjects to CS or UCS (self-observation)

- Important: predictive significance of the CS in heralding the UCS (there are certain conditions in which pairing the CS and UCS does not generate the predicted CR)
    - Situational info and prior expectancies give rise to *covariation bias* -- gives distorted perception of covariation
    - Individuals are more prepared to associate certain stimuli with trauma (snakes and spiders)
    - Source of expectancy biases remains unclear (pre-wired due to natural selection?)
- Latent inhibition: when the CS is presented alone before conditioning, it is harder to condition an association between CS and UCS than if the CS hadn't been presented lone before
- Blocking: if UCS is already predicted by CS(A), then signaling the UCS with CS(A) and new component CS(B) results in no learning about new component B

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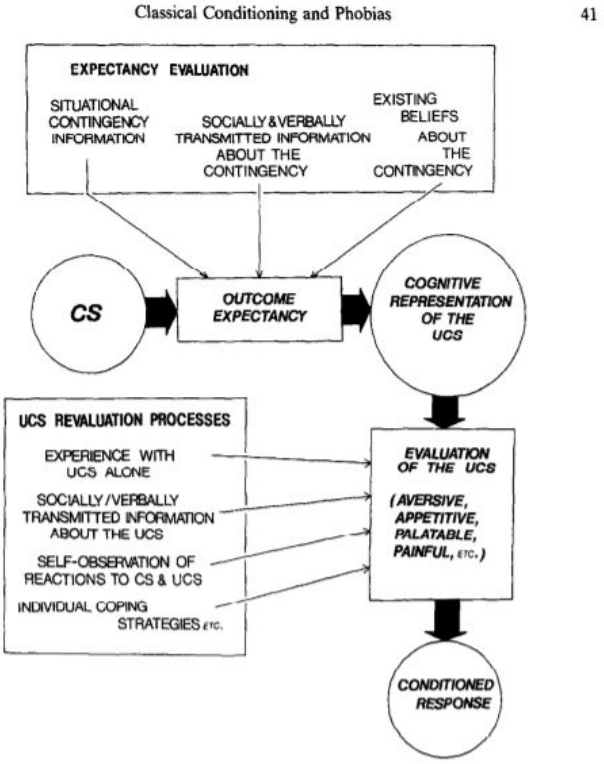
    - Happens more reliably in animals than humans
- Sensory preconditioning: when exposed to 2 neutral stimuli, there are no behavioural changes. When CS2 is paired with UCS, then presentation of CS1 will be enough to elicit the CR appropriate to the UCS (CS1 and CS2 are learned to go together)
    - Called behaviourally silent learning

Model of Human Conditioning

- Processes of contingency learning in humans are way more varied than those in animals (e.g. learning about extinction can produce an immediate dramatic reduction in CR strength)
- We can consider human contingency learning in terms of expectancies
    - Include situational information, social/verbal transmitted info, and existing beliefs around CS-UCS relationship
- This model assumes the associations between CS and UCS can be influenced by factors other than experienced contingency, and adds performance component that suggests strength of CR is determined by nonassociative factors which influence evaluation of UCS



Traditional Criticisms of Conditioning Accounts of Phobias

- Not everyone who experiences pain or trauma develops a phobia for that situation
    - Latent inhibition allows of failure of CR development
    - Strength of CR depends on individuals evaluation of the UCS
- It is possible for individual to learn an association between a CS and UCS when UCS is non aversive, and then reevaluate and have UCS aversiveness inflated later on
- Fears are unevenly distributed
    - Conditioning accounts of phobias should be considered within evolutionary framework
        - preparedness theory: fears to prepared stimuli exhibit: relatively rapid acquisition, resistance to extinction, resistance to the influence of cognitive factors such as instructions/information, learnt more easily with certain aversive consequences
    - Preparedness effects may also be determined by pre-existing beliefs about the relationship between stimuli and outcomes

Constituency of Conditioning Models

Direct and indirect acquisition processes:

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- Three pathways model of acquisition: normal conditioning, vicarious learning through transmission of information or observational learning
- At least some phobia acquisition occurs vicariously and without direct experience of trauma

Spontaneous Fluctuations in Fear and Processes of UCS Revaluation

- *Denial:* coping strategy that can devalue stressful event (but is it positive in the long term?)
- Other coping mechanisms: (found in those with lower anxiety)
    - Positive comparisons, selective ignoring, devaluing the importance of the event
    - Spontaneous remission of symptoms: can have to do with environment of individual getting better, alleviation of aversive event, or by self observation changing

Scope of Conditioning Models of Phobias

- Panic disorder:
    - Encountering internal/external conditioned cues elicits panic
    - Catastrophic interpretation elicits full blown panic when the sufferer perceives a bodily sensation that might be associated with catastrophic beliefs

Treatment

- Latent inhibition: prior nontraumatic exposure to the CS reduces likelihood of it becoming associated with trauma
    - Expose individuals to dental treatment, interviews etc
- Teach coping skills and positive thinking

Expectancy Learning and evaluative learning in human classical conditioning: affective priming as an indirect and unobtrusive measure of conditioned stimulus valence

Expectancy learning: presentation of CS activates the expectation of real US occurrence in the here and now of the immediate future

- Internal or external cue may become established as CS that leads to panic attack

Merely referential learning: presentation of CS activates a representation of the US, without additionally generating the active expectancy of real US occurrence in here and now of immediate future

- CS makes subject think of US
- *Evaluative conditioning*: observation that mere contingent presentation of neutral stimuli with (dis)liked stimuli changes the valence of the originally neutral stimuli into a (negative) positive direction
    - Doesn't depend on awareness of CS-US pairings, and is not sensitive to occasion setting or modulation, CS-US contingency, extinction
    - Is sensitive to US-revaluation
    - Registration of co-occurrences between neutral and valenced events

How to demonstrate both types of learning within one and the same conditioning preparation?

- Need to test expectancy learning as well as evaluative conditioning -- assess verbal ratings and evaluative changes (nonverbal motor or physiological responses)
    - Affective priming task: assess the valence of the CS+ in a way that is unobtrusive and cannot be biased by demand effects

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Discussion

- Method: used differential aversive conditioning preparation with pictures of human faces as CSs and an electrocutaneous stimulus as US
- Repeated contingent presentations of previously neutral face (CS+) and aversive electrical stimulus (US) altered meaning of CS:
    - CS+ became valid predictor for the US
    - CS+ became more negative stimulus
- Experiences that give rise to US-expectancies also give rise to changes in global affective evaluations of the CS

Safety behaviours preserve threat beliefs: Protection from extinction of human fear conditioning by an avoidance response

Exposure with safety behaviour allowed (social phobics allowed to avoid eye contact etc) leads to smaller gains than exposure without safety behaviour

- Similar to protection from extinction
- CS presented with US, then extinction happens when CS is presented without US

Protection: when extinction is impeded by concurrent presentation of inhibitory CS (predictor of absence of US) during extinction phase

Record both skin conductance (objective anxious arousal) and ratings of shock expectancy (self report measure of learning)

Experiment gives clear evidence for protection from extinction of pavlovian fear CS by instrumental avoidance response

- Participants in protection group showed little extinction
- Danger signals and safety signals can yield a protective effect
- Conditioned responding can be cognitive mechanism, due to role of attention, reasoning, and conscious awareness (so may be high level instead of low level)

Renewal and Reinstatement of Fear: Evidence from Human Conditioning Research

What are the determinants of a relapse?

- Return of fear: reappearance of fear that has undergone extinction, does not have to be back to original level, but at least partially

Exposure and Extinction

- CS repeatedly presented without US ➔ decrease conditioned responding (however this is not unlearning the connection)
    - Additional learning - CS no longer good predictor of US, so a CS -no US link is created, inhibiting the CS-US link
    - There can be spontaneous recovery, disinhibition, reinstatement, renewal of CS-US link

Underlying Mechanisms of Extinction

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- Exact information about circumstances of acquisition and extinction is of crucial importance
- In clinical studies: use human fear-conditioning paradigm, most closely resembles real life fear situation
    - Neutral visual stimuli used as CS+, and electrocutaneous stimulus or loud aversive noise is used as US(presented with a CS-)
    - Associative learning effects measured by increase of decrease in differential responding to CS+ and CS-
    - At the end of extinction CS+ has lost US expectancy (an aspect of the fear)

Renewal of Fear

- Most frequently: ABA renewal (testing in original acquisition context after extinction in different context)
- ABC renewal: after extinction in different context, transfer of extinction is tested in a third context
- AAB renewal: acquisition and extinction take place in identical context, but testing executed in new context (least robust renewal)
- Exposure effects are not only restricted to exposure context but also to the specific stimuli used (room exposure treatment is done in, and types of spiders used)
- Acquisition easily generalizes to new context, extinction does not

Mechanisms of Renewal and Clinical Implications

- Extinction information is only effective in presence of exposure-therapy context - generality and permanence of the effects of exposure are pessimistic
- Extinction context directly predicts non occurrence of US and thus protects the CS from extinction
- Or fearful object is perceived as partially different stimulus in exposure context than in acquisition context
- Will be helpful to imitate the original acquisition situation whenever possible during treatment

Prevention of Renewal

- Use retrieval cues: stimuli that can be carried from extinction context to test context
- Extinction in multiple contexts: test in original acquisition context may produce less renewal than after extinction in only one context
- Manipulating contexts can effectively reduce the fear of spiders

Mechanisms that prevent renewal of fear and their implications

- Direct inhibitory association model: multiple contexts increases the number of safe contexts that develop an inhibitory association with the US
- Modulatory mechanism: number of contexts in which CS is no longer followed by US increases
- Multiple context method may have impact on learned association and therefore may change the CS-US acquisition rule

Reinstatement of Fear

- Return of extinguished conditioned responses caused by the experience of one or more US-only presentations after extinction

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    - Control groups that do not receive the additional US-only trials often have a partial return of fear
- Confrontation with unpredicted US's after extinction can restore part of the original fear

Mechanisms of Reinstatement:
- Does reinstating US need to be the same US as the one during acquisition?
    - can also be a different US than the one from acquisition -- but shares affective qualities
    - Reinstatement can be reduced by exposure to the reinstatement context after the presentation of the reinstating

Exposure Treatment in multiple contexts attenuates return of fear via renewal in high spider fearful individuals

Renewal of fear may occur when feared stimulus is encountered outside of the treatment context
- Conducting exposure treatment in multiple contexts attenuates fear renewal
- Treatment using multiple stimuli helps generalisability of treatment

Study done to examine whether conducting exposure treatment in multiple real life contexts with a real life spider enhances the generalisability of exposure treatment to novel contexts and attenuates renewal of fear
- Participants allowed to complete any step they were willing (move freely within contexts)
    - Done to measure avoidance, heart rate measured for fear
- Control group: receives treatment in one context, follow up in same context
- Single extinction context group: treatment in one context, follow up in novel context
- Multiple exposure context group: treatment in 3 different contexts, each follow up in different context

Results show renewal of fear can be attenuated by conducting exposure treatment in multiple real life contexts
- Different contexts and different environments for the spider focused the participant on the procedure and less on the fact that it was an experiment
- Spider phobia chart used

Pavlovian Influences over Food and Drug Intake

When individuals are able to predict accurately that a bodily parameter is going to be altered by an external event, they can learn to initiate a conditioned response in anticipation of the perturbation that minimizes its impact.

Regulatory Implications of Food and Drugs
- Through pavlovian conditioning, we are able to maintain desirable aspects of good and drugs, while minimizing less desirable negative effects of each

Biological Consequences of Meal Taking
- Meals initiated when glucose utilization reaches low threshold (simple depletion-repletion model)
- However:
    - Consequence of eating -- post prandial hyperglycemia

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- This is stopped by food-associated cues, which elicit insulin secretion (activation comes from the brain, from the food stimuli)
- When arbitrary stimuli are reliability associated with food, they initiate insulin secretion via vagal pathway from brain
    - Blood glucose is lowered, and metabolic rate and body temperature change
- Animals can adapt to eating several small meals or few large meals a day -- if onset of meals is predictable
    - If the environment is not regular, of if adaptive responses are prevented from occuring, animals can not tolerate large meals

Impact of drugs on regulatory systems

- Drugs can cause effects that interact with ongoing neural regulatory mechanisms
- Like food, it alters parameters that the body normally monitors and controls
- Tolerance can occur due to a conditioning mechanism
    - Environmental cues (CS) ➜ neural response of drug effect (UR)
    - Perceived disturbance of regulated parameter caused by drug effect is US
    - CR can be elicited from CS without presence of drug (conditioned)
    - In absence of CS, but still getting the drug the individual will not seem tolerant
        - When stimuli signal that drug is coming, will be tolerant
        - Specific association is necessary, it doesn't matter how many times the drug has been received
    - If individual presented with CS but no drug (placebo), body with elicit the CR response but without the drug competing with it, there is the withdrawal symptoms

Differences food intake and chronic drug use

- Development of behaviour tolerance is unavoidable consequence of being able to learn anticipatory responses to homeostatic perturbations
- Learning plays important role in drug addiction process - there are parallels to this process in eating

Learning Model of Binge Eating: Cue Reactivity and Cue Exposure

Psychological mechanisms responsible for binge eating??

Cue Reactivity

- Elicits strong bodily reactions (in drug addicts: increased heart rate, salivation, skin conductance in response to drug related cues)
- Cues that are almost always and exclusively present when drug is being administered will acquire ability to predict administration and effects of the drug
- Classical conditioning models of relapse: after systematic association of environmental/interoceptive cues with drug administration, the cues reliably signal the drug effects
    - When cues are good predictors of administration, they can elicit the responses and cravings which increase likelihood of drug use

Binge Eating and Cue Reactivity

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- Mere anticipation of food (senses) and cognitive processes (thinking about food, hypnosis etc) elicit responses which prepare organism for digestion of food - insulin release
- Food is US, metabolic responses are UR. Cues that signal food intake are CSs which trigger cue reactivity (CRs)
- Learning model of binge eating -- offers explanation for the large collection of disinhibitors identified in binge eaters
    - Many cues can stimulate binge eaters to overeat
    - These all act as conditioned stimuli with identical response: binge eating

Predictions

- Classical conditioning of food intake to exteroceptive or interoceptive stimuli will result in cue reactivity
- After a variety of disinhibitors binge eaters will show cue reactivity
- Inducing cue reactivity will be experienced as urge to binge
- Treatments that fail to reduce cue reactivity will have higher rates of relapse than treatments which are successful in the reduction of cue reactivity

Cue exposure with response prevention: Practical aspects

- Cues will elicit reactivity as long as they remain reliable predictors
- For cue exposure, subject should be exposed to cues but prevented from usage (substance use usually)
- During exposure cues are presented, craving is made as strong as possible, and eating is prevented (in vivo as much as possible - in the home of patient etc)
- Flooding: presenting as many cues as possible and starting difficult helps most with extinction
- If there is cognitive avoidance therapy is stopped
- Therapist moves away from scene as quickly as possible (usually after 3-4 months)
- 45 minutes session approximately -- extinction within 6 sessions
- However: subjects who were given opportunity to leave when anxiety was very high also profited from exposures as much as those that stayed in the situation
- Sessions need to be repeated regularly at short intervals
- Small amounts of the binge food are eaten during the therapy in conditions that will not cue a binge

Cue exposure with response prevention: Empirical status

- Cue exposure studies show large reduction in subjective craving
- Most positive findings from alcohol exposure studies
- Exposure had better results than avoiding stimuli studies
- Do not combine these methods with antidepressants, as those reduce cue reactivity
- May lead to anorexia! Need to be careful

Increasing Efficacy of Cue Exposure Treatment in Preventing Relapse of Addictive Behaviour

From contemporary learning perspective

CERP: Cue exposure with response prevention

- Drug addict repeatedly exposed to stimuli associated with addictive behaviour
- Extinction of cue reactivity often does not generalize beyond treatment setting

Theory:

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- Extinguished conditioned responding can recover some time after extinction = spontaneous recovery
- Renewal effect = when CS is presented in a different environment than where the extinction took place
- Conducting CERP in only once place is limiting

Multiple contexts CERP
- Enhances probability of generalization of extinguished cue reactivity to an environment other than the treatment setting
    - Long process, still does not prevent spontaneous recovery

CERP with retrieval cues
- Salient features of the extinction environment, facilitating the inhibitory meaning of the conditioned stimulus even outside the extinction context
- Still unclear what is best retrieval cue -- clothing, reminder card?
- A good alternative to lengthy multiple contexts but still needs research

Lecture 4: Associative Learning
US ➜ UR
CS ➜ UR

CS ➜ CR

US revaluation
- US without CS, same characteristics but creates different response
- What is learned is a predictive relationship: predictive value of CS ➜ anticipation

Example anticipation
Always coffee at breakfast ➜ blood pressure up
- Caffeine = US
- Preparations in morning to take coffee = CS
- Blood pressure = UCR

Unaware of decaf at breakfast ➜ what happens to blood pressure?
- The US is absent
- Blood pressure goes down (you feel even worse than before)
- Conditioned compensatory response: blood pressure lowers as compensator for the expected increase
- If you were aware that is was decaf your blood pressure would not go down (but there might still be some compensatory response)

Example Placebo
Ethanol ➜ hypothermia
CS1 (green syringe) -- ethanol
CS2 (brown syringe) -- placebo
Placebo in green syringe ➜ body temperature will decrease, has to compensate for expected hypothermia

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Conditioning and Addiction

Drug Cues: predictor drug intake → compensatory response (CCR) body preparing itself → is experienced as craving → misuse

- Highly context dependent (internal/external)
- Strongest CCR for stimulus that is always and exclusively associated with intake

Clinical implications?

Modify predictive value of drug cues

- CERP (Cue exposure with response prevention)
    - Relevant context - cue - no intake
- Elicit proper internal and external context
- Need to use multiple contexts and retrieval cues

ABA renewal: learn in one context, detox in another, then when in learning context again there is symptoms renewal

Binge eating: conditioning Pathway??

- Ritualised conditions
- Predictable disturbance of homeostasis
- Cues → insulin production → lowered glucose → craving → misuse

Food cues: predictor food intake → CCR → craving → binge

- CERP:
    - Relevant context (cue → no eating)
    - Eating in absence of cues (no cue → eating)
- 8 exposure sessions until binges are halved (on average), and at 6 month follow up almost no binges

Anxiety Disorders

- Unrealistic, uncontrollable fear of particular stimulus
- Etiology: CS(previously neutral (car)) + UCS (intrinsically aversive (accident)) = UCR
    - CS → CR
    - Harm avoidance
- Phobias often occur because of self experience of negative experience, seeing others experience it, learning about it
- Why do UCS expectancies persist?
    - Illusory correlations (covariation bias) -- type of confirmation bias, illusion of a negative outcome that is untrue
    - avoidance / safety behaviours
- Translational approach: treatment implications:
    - Violate the predictive relationship!
    - Expose to fear elicitors, with response prevention (no safety behaviour allowed)

CS- preexposure → latent inhibition

- Low fear adults: have high familiarity with dogs before aversive encounter
- High fear adults: have low familiarity with dogs before first aversive encounter

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Problem: return of fear

- Associations are forever
- You only get additional learning, no unlearning
- You should try to get extinction in as many contexts as possible

Notes Week 5

Interpersonal Processes in Social Phobia

Interpersonal disorder: condition in which anxiety disrupts individual's relationships with other people

Interpersonal perspective

- Self-perpetuating interpersonal cycle
    - People that expect others to respond positively to them engage in social behaviours that elicit favourable responses and vice versa
    - Relationships not only our habitual social behaviour, but also sense of self and others

Social Anxiety Disorder and Social Relationships

- Obvious: people with social phobia have fewer social relationships than other people -- friends, dating, less likely to marry
- Patients with partners report greater life satisfaction than those without
- Lower levels of social and emotional intimacy and more negative and blaming towards partners
- Avoidant,non-assertiveness, over-reliant on the relationships they do have

Self-perpetuating Interpersonal Cycles

- Behavioural Patterns
    - Can appear less skillful and more anxious than other people
    - Non Assertiveness, anxiety, withdrawal
    - More state and trait anger
    - Angered when criticized
    - Avoidant personality disorder = severe social anxiety and avoidance -- predicts assault and spousal murder
- Others reactions
    - Shy individuals rated more negatively on variety of interpersonal dimensions, and seen as less intelligent
        - Longer exposure = more positive about socially anxious individual
    - Struggles with socially anxious peoples inability to reciprocate self-disclosure (seem disinterested)
    - Moody, self-pitying, irritate and alienate others
    - "It may be ok to be anxious as long as you're friendly"

Social Skill Deficit or Self-Protective Strategy

- Socially anxious people do not always display avoidant or maladaptive social behaviours
- Dysfunctional behaviour is result of cognitive and emotional processes that are activated by social cues

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- When social conditions are right, they appear capable of behaviour that elicits positive interpersonal response

Cognitive Processing of Social Information
- Social schema and the self
    - Anxiety arises when person becomes aware of discrepancy between knowledge structures representing actual self and ought other self
        - Social anxiety ppl have larger discrepancies
    - Very sensitive to criticism/disapproval
    - Activation of negative social information activates negative self-information
- Selective Attention to Social Cues
    - Selectively process threat-related information: internal (anxiety) or external (cues from other people)
    - Attentional bias toward threat information followed by immediate avoidance
- Interpretation of Social Cues
    - Interpretation bias in self-related judgments and others responses to own behaviour
    - Possible negative biases in social judgments of shy people

Social Pathogenesis
- Development studies show that hypersensitivity to environmental change operates to inhibit behaviour -- inhibited behaviour has been shown to predict social timidity
- Social anxiety can be produced by adverse social experiences even in children who are not initially inhibited
    - Late onset social shyness more likely to report parental abuse

Self Perpetuating Cycles
- Parenting affects childs social anxiety, and childs social anxiety evokes distinct parenting behaviour (bidirectional relationship)
- Parents prefer socialization strategies that limit opportunities for child to develop independence
- Insecure attachment to mother predicts social inhibition!!!

Variability in social learning experiences
- Parents of those with social phobia (either causing or in response to phobia): overprotective and controlling, hostility and abuse, or lack of family socializing
- Emotional abuse and neglect in childhood significantly higher
- Different early social environments may create different developmental trajectories to social phobia
- Adverse peer experiences:
    - Bullying, rejection, neglect
    - Many adults with social phobia can identify traumatic event in adolescence

Interpersonal Processes in Treatment
- Warm category: fear of offending or disagreeing: desire to remain in contact
- Cold category: emotional detachment and hostility
- Warm group does benefit from CBT, cold does not - bodes poorly for treatment outcome
- Can those with social phobia collaborate with therapist??

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    - Not correlated to CBT response, but can create significantly greater change in one on one CBT
- patient -therapist relationship
    - Dynamic interpersonal: help patient realize how biases in their expectations and interpretations of others behaviour lead them to engage in maladaptive behaviours
    - Supportive empathic therapist: studies only show that a bad relationship encourages patient drop out

Interpersonal Subtypes in Social Phobia: Diagnostic and Treatment implications

Use Interpersonal Problems Circumplex scale (IIP-C)

- 8 subscales: domineering, intrusive, overly nurturant, exploitable, nonassertive, avoidant, cold, vindictive
- Can compute scores on each axis, to define location of predominant interpersonal problem pattern

Pathoplasticity

- Mutually influencing, non etiological relationship between psychopathology and another psychological system
- Maladaptive self concepts and disturbed interpersonal relations are key elements of phenotypic presentation of psychopathology

Hypothesis of this study: socially phobic patients with friendly exploitable submissive interpersonal problems would respond more positively to psychotherapy than socially phobic patients with avoidant, cold, hostile interpersonal problems

Discussion:

- There are two distinct subtypes of socially phobic patients
- Distinct interpersonal problems are not due to other moderators (such as gender etc)
- Should research how psychotherapy techniques can effectively target both the maladaptive behaviour of friendly submissive and cold submissive social phobia patients

Depressive Disorders and Interpersonal Processes

Social Skills Deficits and Depressions

- Interpersonal reactions to depressive behaviours start out sympathetic but eventually become rejecting (initial stance in behaviour therapy)
- *Self reports*
    - Evaluate own social skills more negatively than nondepressed people (but more realistically)
    - Depressive realism effect
- *Peer ratings*
    - Rate them as having lower social skill - there is something in the social behaviour of depressed persons that is detectable
- *Behavioural analysis*
    - *Eye contact:* people with depression make less eye contact with conversational partners. Gaze avoidance often precipitated by negative feelings towards self

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- *Facial expression*: less animated and spontaneous expressions (exaggerated sad reaction though)
    - Controls show a happy expression when no instructions are given, depressed people no happiness
    - Try to exert control over facial expression (will change genuine smile into sad one)
    - Also cant assess happiness in others that well (and elevated propensity to see anger or sadness in otherwise low-intensity faces)
- *Posture and gesture:* less gesturing and head nodding
    - Less use of illustrators (gestures that accompany speech in expressive fashion)
- *Paralanguage:* depressed people speak slower, less, and have more and longer pauses, speak with less volume, more monotonous
    - Can only convey sadness with skills
- *Language and Speech conten*t: often negative verbal content, focus on emotional and less constructive in problem solving. Engage in negative self-disclosure
- *Associations between social skills deficits and depression*
    - Social skills lower ➔ worsening of depressive symptoms
    - Or poor social skills are consequent of depression
    - Scar metaphor: people that had depression demonstrate self rated social skills lower than those of never depressed controls, even 2 years after depressive episode had lifted
    - Or poor social skills (and stressful events) are cause of depression

Interpersonal Responses to Depression

- Coyne theory: depressive behaviour puts burden on other people, who get tired of it and give back non genuine reassurance, which depressed person is aware of
- *Excessive reassurance seeking*
    - Major factor prompting rejection is annoying interpersonal style of seeking reassurance
    - Somewhat unique to depression
    - Vulnerability factor
- *Emotional contagion*
    - After talking with depressed patients, controls are more depressed, anxious, and hostile then those who talk to nondepressed patients
    - Target person's depression predicts development or worsening of depressive symptoms/mood in relational partner
- *Interpersonal Rejection of People with Depression*
    - Can be that depressed persons seek negatively valenced feedback from other people to confirm negative views of self

Family Relations and Depression

- *Family of Origin Experiences and Risk for Depression*
    - Risk factor: low care and high protection (affectionless control)
    - Family processes:
        - Feeling ones role in family is devalued
        - Harsh and punitive parenting
        - Negativity and criticism

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        - And other emotional abuse -- this becomes traitlike, so that child thinks it is a part of their character (will blame self for everything)
            - Greater risk for depression than physical/sexual abuse
            - Cognitive vulnerability for later depression
        - Opposite of emotional abuse = adaptive inferential feedback (help people feel better in presence of negative events by stressing specificity and irrelevance to self-worth)
        - Physical abuse: significantly associated with depression, even 40 years later
        - Sexual abuse: 2-2.5 times greater risk of depression than those who were not abused
            - Can see selves as damaged goods
- *Family of Orientation Experiences and Depression*
    - Relational difficulties with spouse and/or child
    - Depression and marital distress often go hand in hand
    - Members of family may contribute to and maintain another's depression
    - Young depressed people get married hastily
    - Wives depression follows decrease in marital satisfaction,husbands have initial levels of depression which lead to marital dissatisfaction
        - Women more vulnerable than men to have depression after marriage is no longer satisfying
    - Problems in role as parent
        - Disengagement from child and negative behaviour towards it
        - Children of depressed mothers often also have depression

Possible Role of More positive social behaviour in the clinical effect of antidepressant drugs

Serotonin and Social Behavior in Animals and Humans

- Serotonin has inhibitory effect on aggression
- Which increases positive social behaviour
- People are generally unaware that their social behaviour has changed
- Tryptophan (serotonin enhancer) probably has direct effect on social behaviour

Effect of Antidepressants on social behaviour in healthy humans

- Given paroxetine or placebo - paroxetine decreased irritability
- Also tried with other antidepressants
- Modest support for idea that antidepressants may decrease agonistic and increase social behaviours in humans

Effect of Antidepressants on social behaviour in patients

- Irritability and anger attacks decrease while on antidepressants
- Significant increase in extraversion in patients treated with paroxetine then those with placebo
- Other drugs used for other disorders also lower anger
- Patients with elevated irritability may respond more quickly to treatment with SSRIs than only patients with depressed mood

Social Interaction during Depression

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- Deficits in social skills, seek reassurance and negative feedback, exhibit interpersonal inhibition and dependency
- Often exhibit irritability and anger
- Higher hostility to those who are depressed

Complementarity in social interactions and implications for mood regulation
- Persons interpersonal actions evoke complementary response

Lecture 5: TAKE NOTES STILL

Notes Topic 6:
- Explain how neurocognitive alterations may help explain the effectiveness of pharmacotherapy
- Describe the roles of gene-environment interactions and epigenetic effects on gene expression in the development of psychopathology
- Discuss what non-human animal experiments can contribute to biological theories of psychopathology

Cognitive Neuropsychological Model of Antidepressant Response

- Purely neurobiological models of antidepressant response are limited as they cannot explain how these actions relate to improvement in mood and overall symptomatic relief
- Antidepressants may not be acting as direct mood enhancers?

Role of Negative Affective Biases in Depression

- Negative biases in emotional and reward processing across range of cognitive emotions
    - Perceive ambiguous info as negative (faces, self-descriptions)
    - Associated with activity in limbic and striatal circuitry
        - Reduced activity in amygdala, thalamus, putamen, hippocampus
- Display blunted responsiveness to both anticipated reward and actual reward outcomes, but respond terribly to negative feedback
- Negative affective biases associated with relapse
    - High neuroticism
    - family /personal history of MDD

Effects of Antidepressants on Affective Processing

- (subchronic) 7 day SSRI increased recall of positive self-descriptors and seeing ambiguous expressions as positive
    - All occur in absence of significant changes in mood
- Normalizes hyperactive amygdala, ventral striate and frontoparietal cortical responses to negative affective info
    - Placebos do not do this in long term
- SNRIs enhance reward processing, but SSRIs diminish processing of both aversive and rewarding stimuli (general dampening of all experiences) (emotional blunting)

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Can model explain delayed onset of clinical effect and symptomatic relief?

- Reduction in preoccupation with negative info would improve mood, reduce withdrawal, increase availability of info processing resources for other cog tasks
- Thus reduced negative affective biases create improved social interactions and behaviour

Risk, Resilience, and Gene x Environment Interactions in Rhesus Monkeys

- Why are some individuals more resilient than others?

Development of Individual Differences in Stress Reactivity

- Normal rhesus monkey development:
    - First month - intimate physical contact with mother (establishes strong attachment bond)
    - Second month - explore immediate physical and social envt
    - After this infant will spend increasing amounts of time away from mother (bonds more with peers)
- Excessively fearful monkey development:
    - Leave mother at laterage
    - Low rates exploratory behaviour
    - Reluctant to interact with peers
    - Great behavioural distress when mothers are away (prolonged cortisol elevation)
        - In later life consume more alcohol
    - Significant genetic component
- Excessively aggressive monkey development:
    - Readily respond to rough and tumble play (are too rough)
    - CSF 5-HIAA low (highly heritable)
    - Also consume excessive amounts of alcohol

Effects of Differential Social Rearing Environments

- Peer-only reared monkeys rapidly develop strong attachment-like bonds with one another within days, though these are non/dysfunctional
    - Peers are not as good as mother at providing secure base for exploring/soothing the infant
    - Explore and play less (more fear)
    - More extreme behavioural responses to social separation
- Display same characteristics as aggressive monkeys (who are raised by mother)
- Consume more alcohol later
- Are both more fearful and more aggressive

GxE Interactions

- Found allele variation was associated with significant differences in number of depressive symptoms - BUT ONLY if also had experienced childhood neglect/abuse or were experiencing high stress

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- Could be that good genes protect from bad environment, or that being raised by mother is a buffer for those with the less efficient allele from developing aberrant patterns (good environment can protect those with bad gene)
- Specific early social experiences can alter gene expression!
- Resiliency in rhesus monkeys and humans could be genetic diversity??

Early Life Adversity and the Epigenetic Programming of Hypothalamic-

Pituitary- Adrenal (HPA) Function

- Epidemiology of affective disorders reflects influence of family life on neural development and mental health
- Parental care linked to epigenetic mechanisms that regulate a specific phenotype: stress reactivity (influences vulnerability to psychopathology)

Parental influences on Stress Reactivity

- Physical and sexual abuse increase endocrine and autonomic responses to stress
- Variations in parental care → individual differences in neuroendocrine and autonomic responses/emotional reactivity
    - → depression risk

HPA function and MDD

- Depression associated with higher levels of cortisol
    - Cortisol increase during the afternoon (normally this is when cortisol levels fall)
    - High cortisol levels in evening predict lasting depression
        - Antidepressants - increase glucocorticoid (GR) expression, functions
    - Corticotropin releasing factor (CRF) - expressing neurons and CRF itself is higher in depressed brains
        - Antidepressants reduced CSF levels of CRF

Development adversity and HPA function

- Childhood trauma strongest predictor of adrenocorticotropic hormone (ACTH) responsiveness
    - Or childhood trauma and adulthood trauma combined
- Childhood adversity stably influences HPA responses to stress

Developmental adversity and epigenetic regulation of HPA function

- Lactating rats exhibit differences in frequency of pup licking/grooming (LC)
- This affects development of behavioural and HPA responses to adult stress
- Tactile stimulation derived from maternal licking = critical environmental signal for regulation of hippocampal GF expression
- How does GF expression remain elevated after weaning and separation from mother?
    - May be an epigenetic modification that maintains the effect until adulthood

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Lecture: Interpersonal Theory

Descriptive: behaviours, relationships. Social info, social skill deficit
Explanatory: interactional model, self-perpetuating cycles, interpersonal cycles, self-protective strategies, social learning

Interpersonal situations

- Relate self to others, during which social learning happens
    - Proximal and mental (internalized memories, dreams, expectations)
- Social learning: the continuous repatterning of interpersonal experience
    - Social environment changes over time
- Self perpetuating cycle: certain patterns of behaving, feeling, perceiving, understanding. Interpreting are reinforced

People develop complex strategies to regulate self and others

- Self system: beliefs and assumptions about self in relation to others
- Behaviour: interpersonal messages sent to others
- Affect system: feelings about self in relation to others (and expressions such as facial)

Interpersonal theory assumptions:

1. Most important expressions of personality happen in interpersonal situations
2. Interpersonal situations can be conceptualized using the meta-structure (agency and communion)

Interpersonal Circumplex

- Vertical axis: agency
    - Why: to be powerful
    - Style: dominance
    - Behaviour: assertive
- Horizontal axis: communion
    - Why: to be close
    - Style: warmth
    - Behaviour: friendly
- Complements: vertical attracts the opposite, horizontal attracts the same (these satisfy the interpersonal motives)
- The circumplex can be used to graph interpersonal personality
    - In which quadrant is individual?
    - Interpersonal distress: the more area that the person covers in the graph, the more distress

Social Phobia:

- Social anxiety disorder (SAD)
- Circumplex: high social avoidance, non-assertiveness
    - A lot in quadrant 4 (submissive)
    - Will be dominated by others

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- Dysfunctional behaviour only occurs with certain contextual clues
- Developmental dysregulation:
    - self : lack power/agency
    - Behaviour: high submissive
    - Affect: tense/alert

Depression:
- Cuts of impulses to integrate constructive situations with others
- Also in quadrant 4 (socially avoidant, nonassertive, cold)
- Will be rejected/avoided by others
- Development dysregulation:
    - Self: low agency/communion
    - Behaviour: low dominance/warmth
    - Affect: annoyed, frustrated

Interpersonal Pathoplasticity:
- Interpersonal styles shape presentation and course of symptoms
- Friendly submissiveness is most common in those with SAD/MDD (with higher friendly behaviour than controls)
    - Unassuming, compliant, overly trusting, accommodating, exploitable
    - Value: recognition by others
    - Sensitive to: separation, accusation

Models:
Diathesis stress: interpersonal style → affective symptoms (with stress as a mediator in between)
Transactional: interpersonal style → stress → affective symptoms (with IPS contributing to stress)

Treatment
- patient -therapist interaction is an interpersonal situation
    - We need to first build an alliance with patient
    - Friendly submissive patients are more likely to agree to alliance, not drop out etc
- Therapist can also be mediator: change the interpersonal behaviour of client

Lecture: Biological Perspective
- Pharmacotherapy
- genetics/epigenetics

Depression medication often has suboptimal effectiveness, and delayed onset of therapeutic action
- Biopsych theory: neurocognitive methods

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Biology approach: chemical actions cause a delay in downstream neuroplastic effects which lead to an improved mood

Biopsych approach: chemical actions cause a delay in downstream neuroplastic effects, which lead to an improved mood, but with change in emotion bias as a mediator

Facial recognition test:

- MDD patients → have bias for seeing fear when its not there (fear stimuli are interpersonal stimuli)
- MDD patients with meds → within 7 days less bias to fear

Psychological change is represented in the brain: SSRI treatment normalizes amygdala hyperactivity, meaning there is less fear response

Genes can predict psychopathology → trauma alters brain function

- Serotonin transporter gene (SLC6A4)
- Promoter length can be short or long (a pair)
    - SS
    - SL
    - LL
    - Those with S genotype are slower at transporting serotonin
- Animal studies with this genotype
    - Rhesus monkeys also have SS, SL, LL genotype
    - Can have environment imposed on them (mother taken away)
        - MR = mother reared
        - PR = peer reared
    - Results:
        - PR childhood: high negative emotions, low activity
        - PR adolescence: high aggression, low social competence
        - PR adulthood: high stress activity, high alcohol consumption
        - Clear biological effects in the monkeys that were most affected = that had at least one S allele and PR

For charts see notes in book

MDD

Genetics:

- Genetic predisposition, but only those exposed to adversity actually develop MDD (genotypic)
- But more complex!

Epigenetics:

- Inherited
- Can also occur during the course of life (hormones, environment)
- Focuses on gene expression (phenotypic)

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- Heritable but reversible changes
- Methylation
- How it works:
    - DNA methylation → methyl attaches to DNA to tag it (making it more or less active)
    - Histone modifications → influence DNA wrapping, if less tight = more room for transcription factors

Rats

- High licking and grooming, and arched back nursing lead to a serotonin release
    - Babies have low cortisol levels, low anxiety, and high licking and grooming for their offspring
- Low licking and grooming leads to a non-release of serotonin, transcription factor is blocked
    - High cortisol levels, high anxiety, and low licking and grooming in offspring

Humans

- High methylation of SLC6A4 is associated with a smaller hippocampus and history of abuse
- Environment factors, DNA, and epigenetic factors work together to produce MDD
    - Stress can contribute to psychopathology by causing epigenetic modifications in genes, making them work differently

Scientific Status of Cognitive Appraisal Models of Anxiety Disorder

Cognitive perspective divided into two approaches

1. Emotional disorder results from problematic beliefs and appraisals ascertainable bia introspective self-report
2. Emotional disorder results info-processing abnormalities whose influence is inferred only from overt behavioural indices (not self report)

Appraisal Approach

- Beliefs matter -- problematic beliefs and their resultant catastrophic missapraisals lead to disorder
- Rationalistic: disorder makes sense as soon as relevant beliefs and appraisals are established
    - Method in the madness
- Inspires effective treatments

Critique of Appraisal

- Reliant on introspective self-report (falls outside the boundaries of legitimate science)
- MacLeod - compelled to postulate unobserved info processing mechanisms that mediate between stimulus and response to account for explananda
    - But methods remain behaviouristic

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- In clinical psych, there was a liberalization in method as well as theory
- People are bad at identifying factors that influence their behaviour
    - Always search for a cause
    - Catastrophic thoughts and bodily sensations occuring does not mean there is causality (post hoc fallacy)
- People are also often unaware of causes of behaviour

Reappraising Critiques of Appraisal

- Appraisal approach has had better therapeutic results than the info-processing approach
    - This does not confirm its truth!
- Stays in power because of *folk psychology*
    - Quotidian theory we apply in everyday life to predict and explain behaviour of others
    - Lack of this may be what causes social impairment in those with autism
- Overt behavioral indices are better measurement, but self-report is not wholly disreputable
- Aspects of cognitive science rely on phenomenal awareness
- However, verbal reports are valuable and thoroughly reliable when conducted properly

Conclusion

- Depending on question, a self report can be precisely what is needed
- People can provide answers to what-questions, but not to how-questions
    - What is maiden name of mother? Quick answer
    - How did you access the correct name? No answer

Effect of Single Session Attention Modification Program on Response to Public Speaking Challenge in Socially Anxious Individuals

Relationship between attention bias to threat and social anxiety

- Only correlational

Discussion

- Attention training effective in reducing attention bias to threat as well as anxiety response to a social challenge
    - Evident in self-report measure and observers ratings of the speeches
    - Affects vulnerability to anxiety
- Anxiety is associated with deficits in attentional control
    - Thus improving ability to control attention in presence of threatening stimuli may facilitate processing other types of social info
- This training directs participants' attention away from threatening cues (against principles of exposure?)
    - Avoidance can facilitate the effect of exposure under certain conditions
- This study supports causal role of attentional bias in maintenance of social anxiety

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Restrained Eaters show enhanced automatic approach tendencies towards food

Restrained eaters often fail and indulge in the foods they want to avoid

Approach/avoidance tendencies

- Automatic approach tendencies could disturb normal regulation of automatic affective associations with food

Discussion

- Independent of restrained status, participants showed stronger automatic liking associations with high far food than with low fat food - high fat preference was absent in more deliberate self-reported liking evaluations
- Restrained eaters show relatively strong approach tendencies for food -- motivational aspects with respect to food may play important role in dysregulation of food intake
- Lowered self-reported craving for high fat than low fat food in unrestrained eaters (may protect against overeating?)

Approach Bias Modification in Alcohol Dependence: Do Clinical Effects Replicate and for whom does it work best?

Alcoholism = neurocognitive disorder. Premorbid vulnerability factors interact with neuroadaptations that result from excessive drinking

- Strong reward response plays initial role, then habits from using other mechanisms of brain
- Lack of cognitive control over impulses
- Often starting in adolescence is risk factor, but most clinical patients are in adulthood

New treatment:

- Cognitive Bias Modification (CBM)
- Computerized interventions aim to change automatic cognitive motivational processes
- Train to avoid alcohol

Approach bias retraining may work especially well for patients with relatively weak executive control (EC) capacities

1. Test whether the effect of adding computerized approach bias retraining to cognitive behavioural treatment increases abstinence in alcoholic patients and whether effect on treatment outcome would be mediated by the amount of change in approach bias
2. Investigate whether success of training could be predicted by patients level of EC and background variables

Methods:

- Alcohol Approach Avoidance Task (AAT) - use joysticks to react to pics of alcohol/soda
- Color stroop

Discussion:

- CBM could reduce ambivalence by strengthening avoidance impulses, weakening approach tendencies and control over these tendencies

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    - Easier to influence ambivalent action tendencies than to make already existing tendencies even stronger
- Long term effects:
    - Fewer relapses occurred
    - CBM can have influence on young and older brains
        - Young people often spontaneously mature out of addiction
- CBM is promising treatment add-on for treatment of addiction as well as other psychopathology
    - Older people profit most
    - Which training works best for whom??

<u>Cognitive Vulnerability to Depression: Dual Process Model</u>

Two modes of info processing:

- Associative: quick, effortless processing that rests on well-learned associations (automatic)
- Reflective: slow, effortful processing that rests on symbolic rule based inferences

Cognitive theories of depression:

- Cognitive factors are causal agents
    - Negative self-schema
    - Info processing model

1. Generalist dual process model
    a. How people process info in a quick effortless manner
        i. Associative processing (pattern completion)
        ii. Past experiences facilitates processing of current info
        iii. Preconscious level
        iv. Relies on associations in memory that have been formed with repeated experience
        v. Intuition, gut feeling
    b. How info is processed when person is able to engage in effortful thought
        i. Reflective processing (rule/rational based)
        ii. Intention and awareness
        iii. Quite slow - operates sequentially
        iv. Guided by rules that come from language, media, culture, individual rules
        v. Can use rules that come from a single experience
    c. When is mode of processing is likely to be engaged
        i. Operate simultaneously or in sequence??
        ii. Associative is default mode
        iii. Reflective happens when individual has enough resources for this type and is motivated to do so
            1. Often when our expectations are violated
        iv. Associative processing is dominant until unexpected stimulus is encountered

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Dual Processes and Mood Regulation

- Associative processing used to maintain mood, reflective processing used to change mood
    - When certain affect intensity or duration is reached, this triggers shift from associative to reflective processing -- which then alters and repairs current mood state
    - Mood persistence can thus trigger reflective processing (expectations of how long a certain mood should last are violated)

Dual Process Model of Cognitive Vulnerability to Depression

- Negatively biased self-referent associative processing provides foundation for cognitive vulnerability to depression
    - Person's first thought may reveal vulnerability to depression
- Corrected reflective processing can overcome this!
- But not corrected when
    1. Biased associative processing violates expectancies but cognitive resources are not available to engage reflective processing
    2. Biased associative processing does not violate expectancies and a need for reflective processing is not realized
    3. Biased associative processing violates expectancies but reflective processing does not adequate adjust negative biased associative processing
- If associative processing goes uncorrected it will influence pattern completion of further processing -- accompanied by dysphoria
    - In absence of repair, dysphoric mood state will continue
    - Aso reflective processing that unsuccessfully repairs moods can also reinforce dysphoric mood state
- Dysphoria will deplete the cognitive resources need to engage reflective processing
    - Brings up irrelevant tasks and intrusive thoughts
- Feedback loop between negative self-referent cognition and dysphoria develops -- makes it difficult for person to disengage from negative thinking

Importance of negatively biased self-referent info processing

- Depression vulnerability is conferred when self-schemas contain themes of loss and worthlessness
- Negative self-referent info appears to have important role in conferring depression vulnerability across multiple theories
- Dysfunctional attitudes about self are also linked to vulnerability

Circumstances when associative processing is uncorrected

- Revealed when:
    - cognitive resources are depleted
        - People recently remitted from depression will not differ from those that are never depressed in number of negative sentences formed

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        - But when having to distract self with other cognitive task, they do have more negative sentences
        - Depleting cognitive resources reveal cognitive vulnerability to depression that predicts future symptoms
        - Vulnerability observed when life stress disrupts attempts to correct associative biases
        - Effortful intention to suppress negative thoughts as long as there are resources enough to maintain suppression -- when life events get stressful, these cognitive ways are depleted -- depressive symptoms
    - negatively biased associative processes do not violate expectations
        - Uncorrected associative bias becomes starting point for downward spiral
        - Poor metacognitive awareness: ability to relate more functionally to depressive symptoms
        - Dichotomous thinking -- endorsing absolutist forms of thinking (extremism)
    - reflective processing does not adjust biased associative processes
        - Ruination: turning ones attention towards symptoms and causes and consequences of these symptoms
        - Increases depression severity and longitude
        - Endorse positive beliefs about rumination (using it to find answers about depression)
        - Hopelessness theory - people who characteristically attribute negative life events to stable and global causes expect negative consequences to follow from negative event, and believe that negative events indicate they are unworthy are at risk for depression
- Mood consequences of uncorrected associative processing
    - Depression vulnerability is associated with difficulty regulating negative mood states
- Similarities and differences to other models
    - Dual process model emphasizes interplay between 2 cognitive processes = better explanation
    - Has explicit link to social and personality psychology

New insights derived from dual process perspective

- Different modes of processing can account for discrepancies in the literature
    - Need to assess both types of processing -- is there vulnerability to depression when cognitive resources are depleted?
- Development of cognitive vulnerability
    - Early development experiences shape associative processing, recent experiences shape reflective processing
    - Bias occurs in kids with lots of failure/criticism/maltreatment/peer and parent victimization/emotional maltreatment
    - Emotional reconditioning may be necessary to change associative processing

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- Repeated or chronic episodes to depression are likely to reinforce negatively biased associative processing
    - Associations made when depressed may be particularly influential
    - Kindling hypothesis: increasingly less severe negative events trigger depression
- Cultural beliefs
    - Transmitted with great frequency → bias
    - Both caucasians and african americans have bias against african americans
- Societal messages
    - Girls more likely to question self worth → more negatively biased processing
    - Contributes to gender differences in depression onset

Altering cognitive vulnerability

- Alter experiences so that negatively biased associative processing triggered reflective processing that will adjust
    - Assumes cognitive resources are available for this
- Alter associative processing so that it no longer produces negatively biased output:
    - Alter the pattern-completion mechanism
- Repetitive exposure to new associations is needed to consolidate results of reflective processing to associative processing
    - Change negative thinking when negative things occur: have second thoughts
- Encourage repeated experiences that will help form new associations
    - CBT: encourages patients to engage in activities in which they are likely to succeed

Lecture: Cognitive Perspective

Two approaches:

1. CBT: related to dysfunctional beliefs/appraisals
2. Cognitive science: Informational processing biases

Dysfunctional beliefs

- Dominant views in CBT:
    - Disorder specific beliefs = psychopathology
    - Crucial to modify these beliefs to have treatments
    - Focus on verbalisiable cognitions (explicit)
        - Negligence of unconscious beliefs/associations
- It is RELEVANT to focus on IMPLICIT associations
    - Because there are two systems: associative and reflective
    - Associations happen in response to stimulus, then time/resources/motivation lead to validation by reflective process

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- Associative mode
    - Preconscious types
    - Automatic, intuitive
    - Guided by memory representations
    - Quite stable
    - Automatic, spontaneous behaviour

- More at risk for an new MDD episode the more MDD episodes you have had
    - Can be due to associative mode: strong automatic self-negative associations
    - Strength of implicit self associations are predictive of recurrence of depression and anxiety disorders

- Rule based mode:
    - Conscious intentional process
    - Slow, requires a lot of cognitive capacity
    - Guided by reasoning
    - Strategic, controllable behaviour
    - Used when our expectations are violent and if there is enough cognitive capacity for it (default is associative mod)

- Dysfunctional automatic associations are problematic:
    - Uncontrollable spontaneous emotional response (blushing, seating, negative mood)
    - Insufficient cognitive capacity
    - Dysfunctional reflective strategy (no correction, overgeneralization)

- Questions:
    - Are people with psychopathology indeed characterized by dysfunctional automatic associations?
    - Do these have specific spontaneous behaviours?
    - Is CBT successful in modifying automatic associations?
        - If not, then people are at risk for relapse

How to measure automatic associations?

- Association 2 types of stimuli
    - Implicit Association Test
        - Reaction task (attribute labels: negative/positive)
        - Categorize a word into one of two categories
        - (negative/alcohol, positive/soda -- then switched)
        - Depends on associations how fast you categorize
            - People with social anxiety are slower with social and positive category than with social and negative

Dissociations between implicit and explicit measures

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- Disinhibited eating
    - Why unable to restrict food intake?
        - Associative: craving: take it!
        - Affective simon task (people have to say as quick as possible: lekker or vies)
            - Low caloric and high caloric, presented in portrait/landscape
            - If portrait say lekker
            - If landscape say vies
            - Measure reaction time
    - Successful restriction:
        - Show very little bias in motivational saliency
        - Anorexia nervosa: so many associative experiences with food and no take
        - No craving -- can adhere to restricted food intake

Does CBT modify automatic associations?

- People who follow CBT show reduction
- Lots of exposure therapy: things decline
- Panic disorder: small but significant reduction
- There is evidence that CBT does have influence on automatic associations

1. Disorder specific automatic associations
2. AA's showed predictive value for uncontrollable impulsive behaviours
3. Underscores importance insight in AA for
    a. Accurate conceptualisation disorders
    b. Accurate diagnostics and therapy effect evaluation
    c. Novel, theory-derived complementary interventions

Information Processing Biases

- Disorder → attention, interpretation, memory problems (feedback loop, these feed the disorder)
- Emotional are associated with motivational goals
    - Fear = goal of avoiding harm
- Probe test with spiders as option -- testing speed again

Selective attention for threat (anxiety disorders)
Anxiety disorder→ urge to avoid → selective attention → quick and frequent detection of threat → avoidance

Selective attention for substances (addiction/eating disorder)
Urge to approach → selective attention → quick and frequent detection of wanted S → craving → misuse

Selective attention for body parts (eating disorder/body dissatisfaction)

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- Selective attention for “ugly” body parts
- Dependents: gaze, fixation, pupil dilation
- Where do you look when you look in the mirror -- pretty or ugly body parts?
- People can be trained to be less dissatisfied with certain body parts

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